and soft tissues. It has been found in virtually every organ of the body except the gastrointestinal tract.1 Most patients with primary infection recover without therapy. In patients with severe primary infection, therapy should be considered. In patients with disseminated disease, intravenous amphotericin-B remains the 'gold standard' of therapy. Local irrigation of amphotericin-B may speed recovery.1 The oral azoles are an attractive alternative, though the duration of treatment is much longer.6

The main difficulty in diagnosis is failure to consider coccidioidomycosis especially in areas where the disease is not endemic. There is close similarity in the clinical presentation of coccidioidomycosis and tuberculosis and coccidioidal granulomas are common in both. This confuses the picture in India and Southeast Asian countries with a high prevalence of tuberculosis.

This is the first reported case of coccidioidomycosis presenting as liver abscess. Any patient in Asia or Australia not responding to appropriate treatment for pulmonary 'tuberculosis' or 'amtic' liver abscess should be suspected of harboring this uncommon fungus.

References

Toxic megacolon in a renal allograft recipient with cytomegalovirus colitis

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We report a 35-year-old man, a renal allograft recipient, who presented with toxic megacolon. Segmental biopsies from the colon were consistent with cytomegalovirus colitis. Serum polymerase chain reaction for cytomegalovirus DNA confirmed the diagnosis. He was treated with ganciclovir but, though his abdominal condition improved initially, he worsened later and succumbed to his illness. [Indian J Gastroenterol 2001;20:114-115]

Key words: Polycystic kidney disease

Toxic megacolon is a clinical entity characterized by fever, tachycardia and other toxic features, associated with dilatation of part or whole of the colon.1 The most common cause of toxic megacolon is inflammatory bowel disease, but it is also associated with many infective conditions, which include Salmonella, Clostridium difficile, Yersinia, Campylobacter, cryptosporidia and amebic colitis.2,5 We report toxic megacolon occurring in a renal allograft recipient secondary to cytomegalovirus (CMV) infection of the colon.

A 36-year-old man had been diagnosed to have chronic renal failure as a result of polycystic kidney disease. He underwent renal transplantation three months prior to presentation and was on immunosuppression with prednisolone, azathioprine and cyclosporin. He presented with a 20-day history of ill-localized lower abdominal pain that worsened with food. There was no history of vomiting. Three days prior to admission he noticed minimal abdominal distension, obstipation and low-grade, intermittent fever without passage of blood in stools.

On admission, he was toxic and febrile. General examination was unremarkable. Abdominal examination revealed minimal gaseous distension with ill-localized tenderness over the lower abdomen. Bowel sounds were sluggish. Rest of systemic examination was normal.

Investigations: Hemoglobin 13 g/dL, total leucocyte count 32000/mm with normal differential count. Serum creatinine was 1.9 mg/dL; serum electrolytes were normal. Total bilirubin was 11.2 mg/dL. ALT 57 U/L, albumin 4 g/dL and serum amylase level 188 SU/L. Initial ultrasonography showed fluid-filled loops. Doppler study showed normal flow in the major mesenteric vessels. X-ray of the abdomen at admission did not show any fluid levels; a few colonic loops were seen. The patient was kept nil orally and on intravenous fluids. He continued to have intermittent severe pain and abdominal distension seemed to worsen. He developed high-grade fever. Repeat abdominal X-ray on the fourth day showed dilatation of colon; the largest loop measured 8 cm. CT scan on the same day showed dilated transverse and ascending colon; the descending colon appeared collapsed. Colonoscopy with minimal air insufflation showed multiple, discrete, punched-out ulcers in the mucosa proximal to the sigmoid colon till the cecum.

Segmental biopsies from the colon showed CMV inclusion bodies involving the endothelial cells and stromal fibroblasts. In addition there was diffuse congestion and focal superficial ulceration. PCR quantification test for serum CMV DNA was positive (0.01 meg).

He was started on ganciclovir with which his abdominal symptoms regressed initially. However, his condition deteriorated and he developed altered sensorium and had recurrent
seizures. Negative cerebrospinal fluid cultures and MRI of the brain ruled out infections causing and demyelinating CNS disorders. His condition continued to worsen and he finally succumbed to his illness.

Cytomegalovirus infection is often implicated in the worsening clinical course of ulcerative colitis and as a possible etiologic factor of toxic megacolon. It is a common infection in immunocompromised patients, and has been reported to be the second most common complication in the post-renal transplant situation. This is possibly the first reported case of CMV toxic megacolon in a renal transplant recipient.

The pattern of histological involvement reflects the clinical presentation. Predominant epithelial distribution of inclusion bodies is associated with mild inflammation as compared to those who have predominant endothelial involvement where there is severe inflammation and ulceration. This patient showed evidence of endothelial inclusion bodies and this could possibly explain the severity of presentation.

The diagnosis of CMV colitis can be easily made on the basis of histological characteristics. Amplification of DNA from biopsies can also serve as a tool for confirmation of the diagnosis. Quantitative assay of serum CMV DNA can predict disease. A cutoff of 0.001 mcg had a specificity of 95% for symptomatic disease. The serum CMV DNA in our patient was higher.

The treatment of CMV colitis has been extensively studied in patients with AIDS, and ganciclovir is the drug of choice. We believe the drug brought about clinical remission in our patient. The reason for worsening was not due to worsening abdominal disease. Repeat biopsies to confirm the efficacy of therapy was not possible as the patient was moribund.

Current interest in CMV is largely due to an increase in the number of cases of AIDS and organ transplant. With easy availability of endoscopy, this condition will be increasingly identified. Early clinical suspicion of the condition permits rapid initiation of specific therapy and may prevent death.

References

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Gastric and intestinal lactobezoars

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Two male full-term infants presented with unusual features of lactobezoar. One had gastric disease while the other had small bowel bezoar. The gastric lactobezoar was managed medically while the intestinal one required surgical intervention. [Indian J Gastroenterol 2001;20:115-116]

Key words: Intestinal obstruction, milk curd

Lactobezoar is an unusual condition in which inspissated milk-curd accumulates and causes mechanical obstruction of the gastrointestinal tract. It was first described by Wolf and Bruce in 1959. In 1980, Lemoh and Watt collected 11 cases from the world literature. Since then, at least 16 additional cases have been recorded in English language literature. We report two more cases of lactobezoars with atypical features.

Case 1: A four-month-old boy from Saudi Arabia had bilious vomiting, abdominal distension, constipation, irritable cry and refusal of feeds for seven days. He was born at full term and his neonatal period was uneventful. Since birth, he had been fed with Similac formula (Ross Labs, Ohio, USA). He contains carbohydrate as lactose 100%, non-fat cow milk protein as whey 18% and casein 82%, fat as coconut oil 60% and soy 40%. He weighed 5.5 kg. The abdomen was distended, with visible intestinal peristalsis. There was no palpable lump in the abdomen. Hematological and biochemical work-up was normal. Abdominal radiograph showed features of intestinal obstruction. Laparotomy revealed a firm 8 cm x 3 cm, intraluminal mass causing obstruction of the distal ileum. As the mass could not be knelled, the segment of ileum lodging it was resected and primary end-to-end ileo-ileal anastomosis done. Postoperative recovery was uneventful.

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